7. Dacie JV, Galton DAG, Gordon-Smith EC, et al: Non-tropical 'idiopathic splenomegaly': A follow-up study of ten patients described in 1969. Br J Haematol 38:185-193, Feb 1978
8. Long JC, Aisenberg AC: Malignant lymphoma diagnosed at splenectomy and idiopathic splenomegaly. Cancer 33:1054-1061, Apr 1974
9. Strauss RG, Bove KE, Jones JF, et al: An anomaly of neutrophil morphology with impaired function. N Engl J Med 280:478-484, Feb 1974
10. Repine JE, Clawson CC, Brunning RD: Primary leukocyte alkaline phosphatase deficiency in an adult with repeated infections. Br J Haematol 34:87-94, Sep 1976
11. Luddy RE, Champion LAA, Schwartz AD: A fatal myeloproliferative syndrome in a family with thrombocytopenia and platelet dysfunction. Cancer 41:1959-1963, May 1979
12. Kaplow LS: Leukocyte alkaline phosphatase cytochemistry: Applications and methods. Ann NY Acad Sci 155:911-947, 1968
13. Krivit W: Overwhelming postsplenectomy infection. Am J Hematol 2:193-201, 1977

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Mushroom Poisoning From *Chlorophyllum molybdites*

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THOUGH POISONING from *Chlorophyllum molybdites* is rarely reported, we describe a cluster of three cases of a brief, self-limited gastrointestinal illness associated with ingestion of this fungus. *C molybdites,* previously known as *Lepiota morgani* or the "green parasol" mushroom, occurs in widely scattered geographic areas. In contrast to cases of *Amanita phalloides* or *A muscaria* ingestion, there is almost never a fatal outcome. Our report illustrates two reasons why mushroom poisoning may be occurring with greater frequency: popularity of and confusion with species containing hallucinogens (for example, psilocybin) such as *Psilocybe cubensis* or *P mexicana;* and collection of specimens for consumption by inexperienced persons attempting to eat a "natural" food diet.

Similar symptoms occurred in all our patients. Within one to three hours after eating the fungus, nausea, vomiting, and diarrhea, which was initially watery and then usually became bloody, developed. With supportive therapy, recovery was complete within 24 hours. All three cases occurred within four weeks in the autumn of 1977. In each case the mushrooms were available for identification.

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Reports of Cases

CASE 1. A healthy 46-year-old woman collected several "brown mushrooms" in San Diego's Mission Bay Park. Two hours after eating four of the small, button-like mushrooms in an omelette, she became nauseated and began to sweat, then vomited the meal. She noted several small mushroom fragments in the vomitus. Two dinner companions also consumed the mushrooms, but had no symptoms.

Four hours after dinner, she went to the emergency room of the hospital at the insistence of her friends. She passed three watery stools and vomited clear stomach contents (positive for occult blood). Pupils were 4 mm in diameter and reactive to light. Because of the possibility of *A muscaria* ingestion, she was given atropine sulfate intramuscularly and admitted to hospital for observation. She did not have jaundice, hematemesis, melena, dysuria or previous history of colitis.

On physical examination the patient was anxious, excited, diaphoretic and talked incessantly. Vital signs were blood pressure of 130/70 mm of mercury, pulse 120 per minute, respiration 24 per minute and temperature 35.8° (96.4°F), taken orally. Examination of head, neck, and chest showed no abnormalities. A fourth heart sound was noted on cardiac examination; surgical scars of a previous hysterectomy and left inguinal herniorrhaphy were present. Neurologic examination was normal save for pressure of speech with appropriate content.

Laboratory examination disclosed a leukocyte count of 8,500 per cu mm and a hematocrit of 47 percent. Serum sodium was 141, potassium 3.8, chloride 108 and bicarbonate 25 mEq per liter. Electrocardiogram and radiograph of the chest showed no abnormalities. Examination of two stool specimens failed to disclose any ova or parasites; bacterial cultures did not show enteric pathogens.

On the morning following admission, samples of the mushroom (Figure 1) which accompanied the patient were identified as *C molybdites.* The patient did well and was sent home that afternoon. She returned to the care of her private physician and reported no further symptoms when contacted by telephone a week later.

CASE 2. A 20-year-old woman with chronic

schizophrenia ingested four "small brown spade-like" mushrooms which she found on the lawn of the mental health facility where she had been admitted. Previously, she had used mushrooms found in a different location for their hallucinogenic properties. Two hours after ingestion, nausea, vomiting and diarrhea developed. Five hours after ingestion, the diarrhea became bloody, and she was admitted to hospital for observation. The patient had no history of diarrheal illness at the institution. She had delivered her second child two months previously, and was being given fluphenazine decanoate intramuscularly, trihexyphenidyl, cyproheptadine and ferrous sulfate. She had no previous episodes of hematochezia, melena, hematemesis, colitis, and was not currently using illicit substances.

Physical examination showed a blood pressure of 110/76 and 110/80 mm of mercury without orthostasis, temperature was 36.9°C (98.4°F), orally, and pulse 120 per minute. Examination of head, neck, chest, extremities and heart showed no abnormalities. Multiple abdominal striae were noted. The bowel sounds were hypoactive; the liver and spleen were not enlarged. The neurologic examination showed a withdrawn, moderately obese woman who appeared normal otherwise. Sigmoidoscopy showed pink, nonfriable mucosa without ulcerations or polyps. A Wright-stained smear of the stool showed many bacteria and leukocytes.

Laboratory studies gave the following values: serum sodium was 140, potassium 3.9, chloride 111, and bicarbonate 11 mEq per liter; blood urea nitrogen (BUN) was 12 and creatinine 1.5 mg per dl. Peripheral leukocyte count was 22,200 per cu mm, with 69 percent segmented and 36 percent band forms, 3 percent lymphocytes, 1 percent monocytes and 1 percent basophils. The hemoglobin value was 19.2 grams per dl and hematocrit was 55 percent. An analysis of the urine was remarkable for a specific gravity of 1.024, pH 5.5, protein 3+, numerous leukocytes, moderate bacteria, and numerous coarse granular and fine granular casts per low-power field. Cultures of the urine and blood had no bacterial growth, and two stool specimens were without ova and parasites or growth of enteric pathogens. Radiograph of the chest, barium enema, and electrocardiogram (EKG) showed no abnormalities.

The patient received fluids intravenously and the nausea, vomiting and diarrhea resolved over the next 12 hours. At discharge, 48 hours after admission, abnormalities of the urinary sediment had disappeared, the hematocrit was 43 percent, serum creatinine was 0.6 mg per dl, and no occult blood was found in the stool. Specimens of the mushrooms consumed were identified as *C molybdites* before the patient was discharged from the hospital.

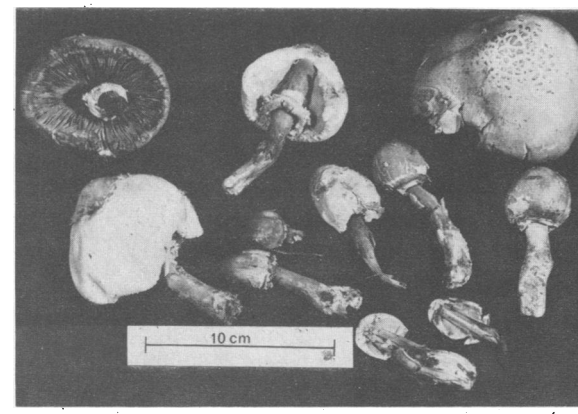

Figure 1.—Various stages of *Chlorophyllum molybdites* ranging from young buttons to fully matured specimens. Only the mature, expanded mushrooms show the characteristic green coloration of gills on the underside of caps due to green spores. Immature spores of younger specimens are white.

CASE 3. A previously healthy 22-year-old man ingested a large, "brown spotted" mushroom found in San Diego's Balboa Park. An hour later he experienced intense nausea and vomiting, yellow colored diarrhea, shaking chills, diaphoresis, salivation, diffuse abdominal pain and diffuse muscle tenderness. He did not have melena, hematochezia or hematemesis. Several hours earlier, the patient had consumed two glasses of red wine and one "Quaalude tablet." He claimed to have previously used similar mushrooms, which he had found in the midwestern United States, for their hallucinogenic effect.

On physicial examination the patient was noted to be shaking and nauseated. Blood pressure was 90/70 mm of mercury supine and 60 systolic in the sitting position. Temperature was 37.7C° (99.9°F) orally, pulse was 100 per minute when the patient was supine and 120, sitting. Head, neck, chest, heart and neurologic examination showed no abnormalities. The abdomen was slightly tender with normoactive bowel sounds and no organomegaly. Occult blood was present in the stool.

Laboratory findings included the following values: hemoglobin 18.6 grams per dl, hematocrit 56.4 percent and 15,000 leukocytes per cu

mm including 28 percent band forms. Serum sodium was 143, potassium 4.2, chloride 105 and bicarbonate 18 mEq per liter. Liver function tests and a radiograph of the chest showed no abnormalities, and an EKG showed only early repolarization. Analysis of the urine showed 3+ proteinuria and 5 to 10 leukocytes, as well as rare leukocyte granular and waxy casts per low-power field. Blood and urine cultures had no bacterial growth.

The patient was treated with fluids given intravenously and supportive care. All symptoms and signs resolved over the next 18 hours. Samples of the responsible mushrooms were identified as *C molybdites*. Analysis of urine and liver function tests gave normal values at the time the patient was discharged, 48 hours after ingestion of the mushrooms.

Discussion

The three patients all had moderately severe, self-limited gastrointestinal illnesses following the ingestion of a common species of mushroom *C molybdites*. While it is possible that these patients had ingested several species of mushrooms, this appears unlikely in view of the following considerations: (1) Only specimens of *C molybdites* were submitted to us by the patients. (2) The areas where the mushrooms were collected by the patients were small and when inspected by us, only *C molybdites* mushrooms were found. (3) These mushrooms grow during a short and limited season and the cases were all confined within a four-week period.

Accounts of *C molybdites* poisonings in the 19th century appear in American and British Commonwealth literature. Perhaps the most illustrative report is giving by McIlvaine and Macadam[1] who quote H. I. Miller of Terre Haute, Indiana:

> Six families here have eaten heartily of them *(C molybdites)*. The experience is that one or two members of each family are made sick, though, in two families, who have several times eaten them no one was made sick. I enjoy them immensely, and never feel the worse for eating them. I doubt if we have a finer flavored fungus.

The authors further quote Professor W. S. Carter of the University of Texas, Galveston, who reported "the poisoning of three laboring men from eating this fungus. They were seriously sick, but recovered." Bessey[2] later mentioned two Michigan residents who consumed green-gilled "shaggy manes" purchased from a local vendor. Vomiting and bloody diarrhea developed in both patients within two hours of eating the cooked mushroom, but both recovered within 24 hours.[2] Cases of *C molybdites* poisoning have resulted in similar gastrointestinal syndromes among immigrant and native Africans.[3,4] The single fatality noted in the literature occurred when a 2-year-old infant died with convulsions 17 hours after eating the raw fungus.[5]

Standard textbooks of mycology generally place *C molybdites* within a nonspecific group of "gastrointestinal toxins." It is widely noted that the fungus appears not to be toxic to all consumers.[1,6-9] A dose-response effect of the poison is inconsistent with the observation that unsusceptible persons may consume large quantities of the fungus without ill effect. Further, because symptoms develop in only a fraction of consumers during an outbreak, geographic variation of the toxin is an inadequate explanation for the variable susceptibility. Because symptoms of perspiration, thirst and giddiness are part of the *C molybdites* poisoning syndrome, it has been suggested that muscarine may play a role in the toxicity.[10] Further speculation has occurred as to the heat stability of the toxin.[4] Many reports, including our first case, are consistent with at least a partially heat-stable toxin. Eilers and Nelson[11] extracted a substance, toxic for mice and chicks, from *C molybdites* found in Florida and concluded that the active principle was a large protein-like molecule, with a molecular weight greater than 400,000 daltons, and consisted of smaller, still toxic subunits of approximately 40,000 to 60,000 daltons. The substance was partially heat labile and subject to proteolytic degradation.

Mature specimens of *C molybdites* are easily recognized by their green spores and gills. No other species among the Amanitaceae bear the spores that lend their green color to the gills on the underside of the cap. The cap measures from 7 to 30 cm, is broad, convex, knobbed or flat. There are numerous scales which are tinged buff to cinnamon in color. The flesh is firm and white. The stalk is 10 to 25 cm long and 10 to 25 mm thick.[1,10] The green spores appear with maturity. Younger specimens (Figure 1) have white gills or are button shaped, and may be confused with the edible *Leucoagaricus rachodes* and *L procerus*.[6] A method of identification of the green spores of *C molybdites* in vomitus and stool specimens has been described, using filtration and microscopic examination for the presence of basidiospores of the fungus.[12] In the absence of the

whole mushroom, this can be a useful method to confirm mushroom ingestion.

C molybdites is common in the warmer midwestern United States, but rarely found in New England. It is seen from New Jersey to Iowa, southwest through Arizona and Texas,[13] and in southern California.[14] The species seems to thrive in warmer climates and occurs in the Philippines, Tahiti, the West Indies, Brazil, Rhodesia[4] and Kenya.[3] Colonies of the fungus are noted to assume a "fairy ring" configuration and are characteristically found in meadows, short grasslands, lawns, and parks after spring and autumn rains.

The green-spored mushrooms *C molybdites* (also known as *Lepiota morgani*) contain a poison which affects some persons yet is harmless to others. In susceptible persons, ingestion of either raw or cooked mushrooms is followed by nausea, vomiting and diarrhea within one to six hours. The diarrhea may turn bloody, and may contain leukocytes. Diaphoresis, tachycardia and postural hypotension may also be seen. Predominance of parasympathomimetic symptoms—pupillary constriction, salivation and lacrimation—are not features of the syndrome and may help to distinguish these cases from those involving *Amanita muscaria*. There is minimal or no fever, and objective abdominal findings are minimal. Leukocytosis may be seen; there may be transitory abnormalities of the urinary sediment including microscopic hematuria, proteinuria and cylinduria. Supportive care, including maintenance of fluid and electrolyte balance should result in complete recovery within 24 to 48 hours. As in all poisonings, it is useful to have specimens of the offending agent available for identification. Observation for a longer period is warranted if *A phalloides* poisoning cannot be ruled out because of the well-known delay in the appearance of toxic manifestations after ingestion of this mushroom.[15,16]

There is renewed interest in gathering mushrooms by inexperienced collectors for both pharmacologic and nutritional use. Physicians may be called to evaluate and treat inadvertent consumption of *C molybdites* as well as more toxic species with increasing frequency.

REFERENCES

1. McIlvaine C, Macadam RK: One Thousand American Fungi. New York, Dover Publications, 1973, p 37, originally published Bowen-Merrill Company, 1900
2. Bessey EA: A case of poisoning with *Lepiota morgani*. Mycologia 31:109-110, 1939
3. Charters AD: Mushroom poisoning in Kenya. Trans Roy Soc Trop Med Hyg 51:265-270, 1957
4. McCarter GRB: Mushroom poisoning in Rhodesia; with a report of a case of poisoning due to *L morgani*. C Afr J Med 5:412-413, 1959
5. Rumack BH, Salzman E: Mushroom Poisoning: Diagnosis and Treatment. West Palm Beach, FL, CRC Press, 1978, pp 36 and 192
6. Duffy TJ, Verger PP: California Toxic Fungi. San Francisco, Mycological Society of San Francisco, Incorporated, 1977, p 23
7. Smith AH: Mushroom Hunter's Field Guide, Revised and Enlarged. Ann Arbor, University of Michigan Press, 1958, p 185
8. Miller OK: Mushrooms of North America. New York, EP Dutton Company, 1972, pp 34-35
9. Lincoff G, Mitchel DH: Toxic and Hallucinogenic Mushroom Poisoning. New York, Van Nostrand Reinhold Co, 1977, p 147
10. Buck RW: Mushroom toxins—A brief review of the literature. N Engl J Med 265:681-686, 1961
11. Eilers FI, Nelson LR: Characterization and partial purification of the toxin of *Lepiota morgani*. Toxicon 12:557-563, 1974
12. Eilers FI, Barnard BL: A rapid method for the diagnosis of poisoning caused by the mushroom *Lepiota morgani*. Amer J Clin Pathol 60:823-825, 1973
13. Murril: Chlorophyllum. N Amer Flora 10:64, 1914
14. Smith CO: *Lepiota morgani* in Southern California. Mycologia 28:86, 1936
15. Wieland T: Poisonous principles of mushrooms in the genus Amanita. Science 159:946-952, 1968
16. Harrison DC, Coggins CH, Welland FH, et al: Mushroom poisoning in five patients. Am J Med 38:787-791, 1965

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Acute Cor Pulmonale Resulting From Tumor Microembolism

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ALTHOUGH lung parenchymal metastatic lesions are relatively common, selective compromise of the pulmonary vasculature due to tumor embolism is rare. This report concerns a case of acute cor pulmonale that developed as a manifestation of diffuse microembolism from previously quiescent metastatic carcinoma.

Report of a Case

A 49-year-old woman was admitted to hospital with a complaint of increasingly severe dyspnea of three days' duration. Five years earlier, surgical excision of a compressive epidural mass established the diagnosis of metastatic adenocarcinoma but resulted in paraplegia at the T-2 level. Subsequently, a primary malignant tumor of the breast was discovered, and left simple mastectomy and oophorectomy were done. In the absence of

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